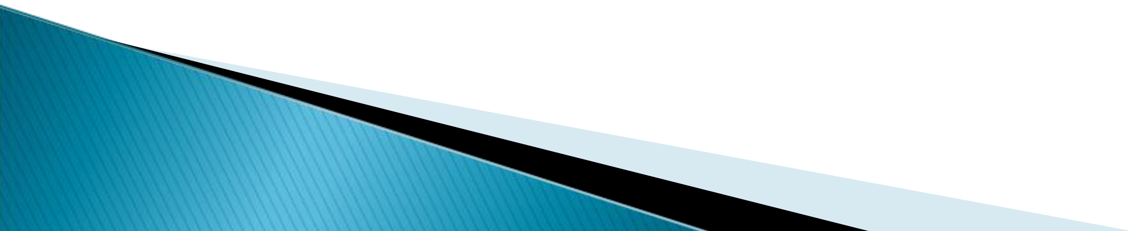
CARDIOGENIC SHOCK



Definition

Clinical condition of inadequate tissue (end organ) perfusion due to cardiac dysfunction

Characterized

SBP < 80_90mmhg lasting > 30 minutes or on intervention support to maintain BP > 90mmgh

PCWP > 15

Evidence of endorgan damage : cool extremities

UO < 30 ml per

(AMS)Altered mental status

serum lactate > 2 mmol

Characterized According to Guidline

***SBP < 80_90mmhg lasting > 30 minutes or onHg inter vention support to maintain BP > 90mmh**

PCWP > 15

Evidence of endorgan damage : cool extremities

UO < 30 ml /h

Altered mental status serum lactate > 2 mmol

Cardiac index < 1.8 and < 2.2 l/min m^2

Genearal accept criteria

- 1. Frank or relative hypotension SBP below 80 or 90 mm Hg or
- reduction on mean arterial pressure (MAP) of 30 mmHg.
- 2. Cardiac index less than 1.8 l/min/m2 without intervention support or less than 2.2 l/min /m2 with support
- 3. Elevated end diastolic pressures on right more than 10 to 15 or left more than 18 mmHg) side of the heart
- 4 evidence of end _organ hypoperfusion

>>Genearal accept criteria

- **Endorgan hypoperfusion may manifest as:**

  -AMS.

  -UO < 30 ml /h. ,

  -serum lactate > 2 mmol

Clinical Trial/Guideline	CS Criteria
SHOCK Trial (1999)[3]	• SBP <90 mm Hg for >30 min or vasopressor support to maintain SBP >90 mm Hg • Evidence of end-organ damage (UO <30 mL/h or cool extremities) • Hemodynamic criteria: CI <2.2 and PCWP >15 mm Hg
IABP-SOAP II (2012)[4]	• MAP <70 mm Hg or SBP <100 mm Hg despite adequate fluid resuscitation (at least 1 L of crystalloids or 500 mL of colloids) • Evidence of end-organ damage (AMS, mottled skin, UO <0.5 mL/kg for 1 h, or serum lactate >2 mmol/L)
EHS-PCI (2012)[5]	• SBP <90 mm Hg for 30 min or inotropes use to maintain SBP >90 mm Hg • Evidence of end-organ damage and increased filling pressures
ESC-HF Guidelines (2016)[6]	• SBP <90 mm Hg with appropriate fluid resuscitation with clinical and laboratory evidence of end-organ damage • Clinical: cold extremities, oliguria, AMS, narrow pulse pressure. Laboratory: metabolic acidosis, elevated serum lactate, elevated serum creatinine
KAMIR-NIH (2018)[7]	• SBP <90 mm Hg for >30 min or supportive intervention to maintain SBP >90 mm Hg • Evidence of end-organ damage (AMS, UO <30 mL/h, or cool extremities)

New stratification of cardiogenic shock By SCAI

A. Or at risk , the patient has risk factors but no signs or symptoms

B. Or beginning ; the patient has clinical evidence of relative hypotension or tachycardia without hypoperfusion

C. Or Classic : the patient present with hypoperfusion without requiring intervention beyond volume resuscitation

D. Or deteriorating : the patient fails to respond to initial interventions

E. Or Extremis; the patient is being supported by multiple interventions and may be experiencing cardiac arrest with ongoing CPR and or extracorporeal membrane oxygenation (ECMO)

Prevalence

Occur in 5 - 8% of STEMI and 2.5% NSTEMI

Mortality

In hospital mortality rate 40 - 60% but with early revascu larization and newer modalities and

aggressive medical treatment provide benefit

Etiology:

1. Acute MI with LV failure 80% or (RVF)
2. Acute MR
3.VSD or free wall rupture
4.sever valvular heart disease(AS , AI , MR , MS)
5. Acute fulminant myocarditis
6. End stage cardiomyopathy
7. HOCM with severe LVOT obstructive
8. Aortic dissection
9. Pulmonary embolism
10. Refractory or prolonged tachyarrhythmia's or brady arrhythmias
11. Post cardiac arrest
12. Cardiac tamponade
13. Drugs

Cardiogenic shock complicating MI

Mostly result from LV dysfunction (80%)

The remainder mechanical defect (VSD,

papillary muscles rupture)

or RV infarction

Rupture of IVS mortality 40_75%

***With transmural infarction**

***Perforation can range from 1 to several centimeters**

***Rupture of the septum with anterior MI tends**

or be apical

***Rupture of septum with inferior MI tend to be**

basal septum and has worse prognosis than

anterior location

Factors associated with increase risk of rapture IV septum
:

1.Lack of development collateral

2. Advance age

3. Female sex

4 . chronic kidney disease

Decrease of likely of septal rupture(due to ischemia induces myocardial preconditioning:

1. **HTN**
2. **DM**
3. **Chronic angina**
4. **Previous MI**

Rupture free wall

* **Most common in the LV specifically anterior and lateral MI and rare in atria .**
* **Mortality 75 -90%.**

Rupture can be associatd with

1. Reperfusion with thrombolytic agent

2. Older age

3. Female sex

4. Single vessel disease without collateral

5. Anterior MI or first MI

Rupture of papillary muscles

Sudden massive MR that develops cannot be tolerated

1. Complicated by transmural infarction.

2. Rupture usually tip or head of papillary muscles .

3. Inferior MI rupture of posteromedial papillary muscles and more frequent than anterolateral papillary muscles.

4. Papillary muscles rupture occur with small infarction.

Rupture of myocardial decrease with PPCI.

Pathophysiology :

***Primary insult is :**

Decrease myocardial contractility



↓ Cardiac output



↓ Hypotension lead



To systemic vasoconstriction



Cardiac ischemia ,peripheral vasoconstriction and end organ damage

>>Pathophysiology :

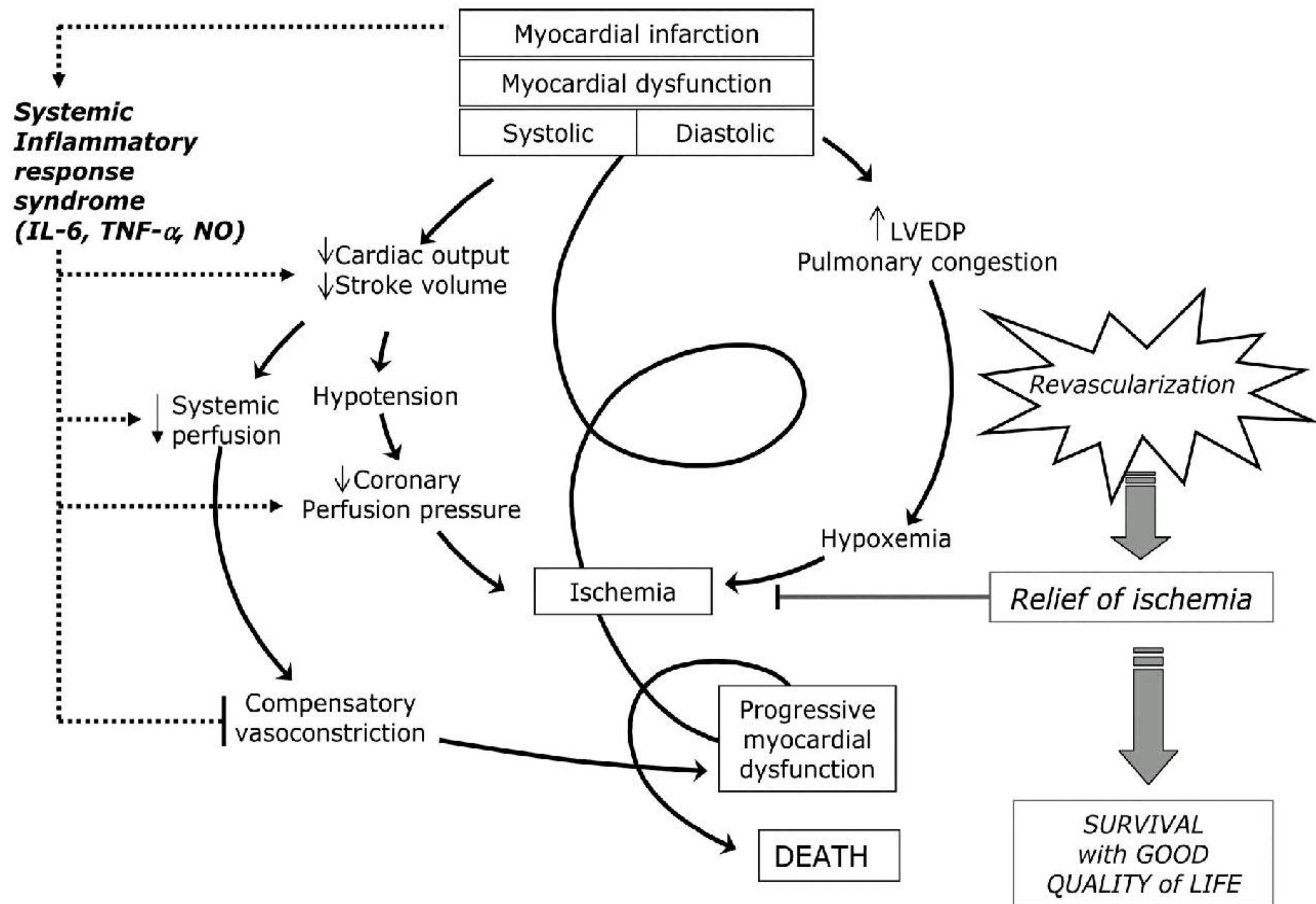
Systemic inflammation causes pathological vasodilation releasing :

Nitric oxide and peroxynitrite which have cardiotoxic inotropic effect

Interlukine and TNF are additional systemic inflammatory mediators that result in vasodilation and increase mortality in CS

RVF result in RV dilatation ,the inter ventricular septum compromising LV diastolic filling and worsening hypotension

Figure 1. Current concept of CS pathophysiology.



Reynolds H R , and Hochman J S Circulation. 2008;117:686-697

>>Pathological finding:

*At autopsy >2/3 of patient with cardiogenic shock demonstrate multi vessel diseased including LAD .

*And exhibit thrombotic occlusion of the artery supplying large region .

*Cardiogenic shock occur with loss of 40%and more of the LV Mass.

*Piecemeal necrosis that is progressive myocardial necrosis form marginal extension of the infarcted into ischemic zone bordering the infarction.

>>Pathological finding:

***Persistent elevation of cardiac biomarkers is due to necrosis of extension of the infarction**

***Extension and focal lesion probably result in part from shock state it self .**

***Early deterioration of the LV function secondary to extension of infarction and ins same case from expansion of the necrotic zone .**

***Hydrodynamic force during ventricular systole disrupt necrotic myocardial muscle bundle lead to expansion and thinning of the akinetic zone result in overall LV dysfunction.**

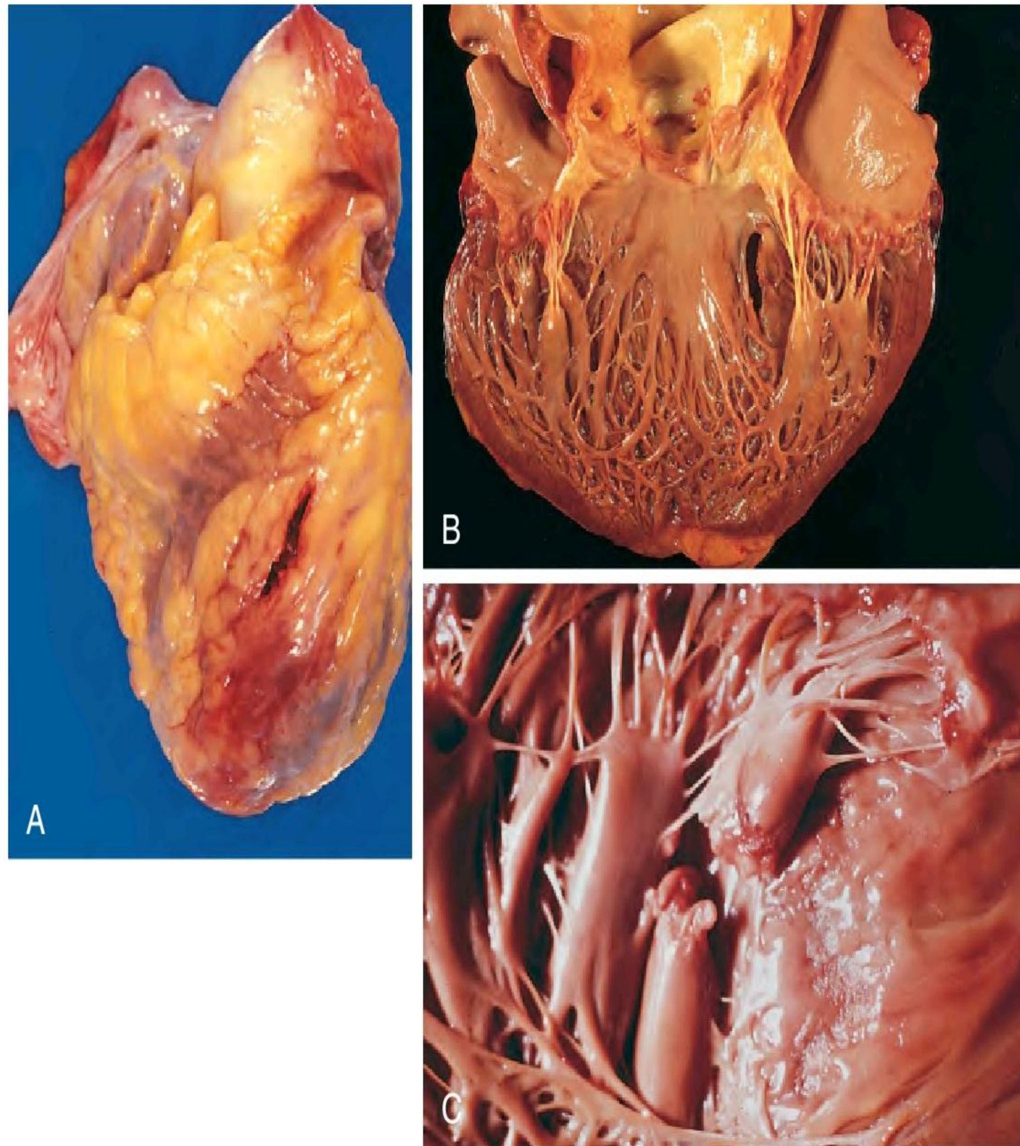


FIGURE 59.27 Cardiac rupture syndromes complicating STEMI. **A,** Anterior myocardial rupture in an acute infarct. **B,** Rupture of the ventricular septum. **C,** Complete rupture of a necrotic papillary muscle. (From Schoen FJ: The heart. In Kumar V, Abbas AK, Fausto N, editors. Robbins & Cotran Pathologic Basis of Disease. 7th ed. Philadelphia: Saunders; 2005.)

Clinical presentation and physical examination

In CS with ACS symptoms and singe and reflect infarction involving 40% of the LV Mass.

***Altered M S.**

***Hypotension.**

***Arrhythmia.**

***Diminished pulses.**

***Dyspnea.**

***Peripheral edema.**

***JVP increased.**

***Orthopnea.**

<<Clinical presentation and physical examination

*Patient with CS most commonly present with cool extremities and singe of pulmonary congestion (termed cold and wet) which reflect reduce cardiac index (CI) ,increase SVR and increased PCWP

*Patient may present euvolemic or dry and cold indicates a reduce CI , increased systemic vascular resistant and normal PCWP

*An under recognized presentation of CS is the wet and warm subtype. And this represent systemic inflammatory response syndrome reaction in conjunction with MI and sepsis and associated with higher incidence of mortality and indicate : 1-Reduce CI

2-Low to normal SVR

3-Elevated PCWP

When should be suspect systemic inflammatory response syndrome ?

Fever

Elevated WBC

Low SVR

25% present of patient with STEMI with systemic inflammatory response syndrome

Initial investigation

- Cardiac catheterization in definitive Dg investigate and guide therapeutic intervention in MI complicated by CS.
- CS is a clinical Dg and no investigation should delay emergent cardiac intervention

ECG within 10 minutes of presentation.

ECG divided in 3 group:

1. STEMI
2. Non STEMI
3. Non ST segment deviation

Normal ECG may not exclude

VT VF ,AF, AVB

Routine investigation

ECG. ABG ,CXR ,CBC ,electrolyte , KFT Troponin, CPK,CKMB ,

Echocardiography , (NT-pro BNP i elevated in decompensated heart failure if it, increased in CS and ACS indicate increase mortality.

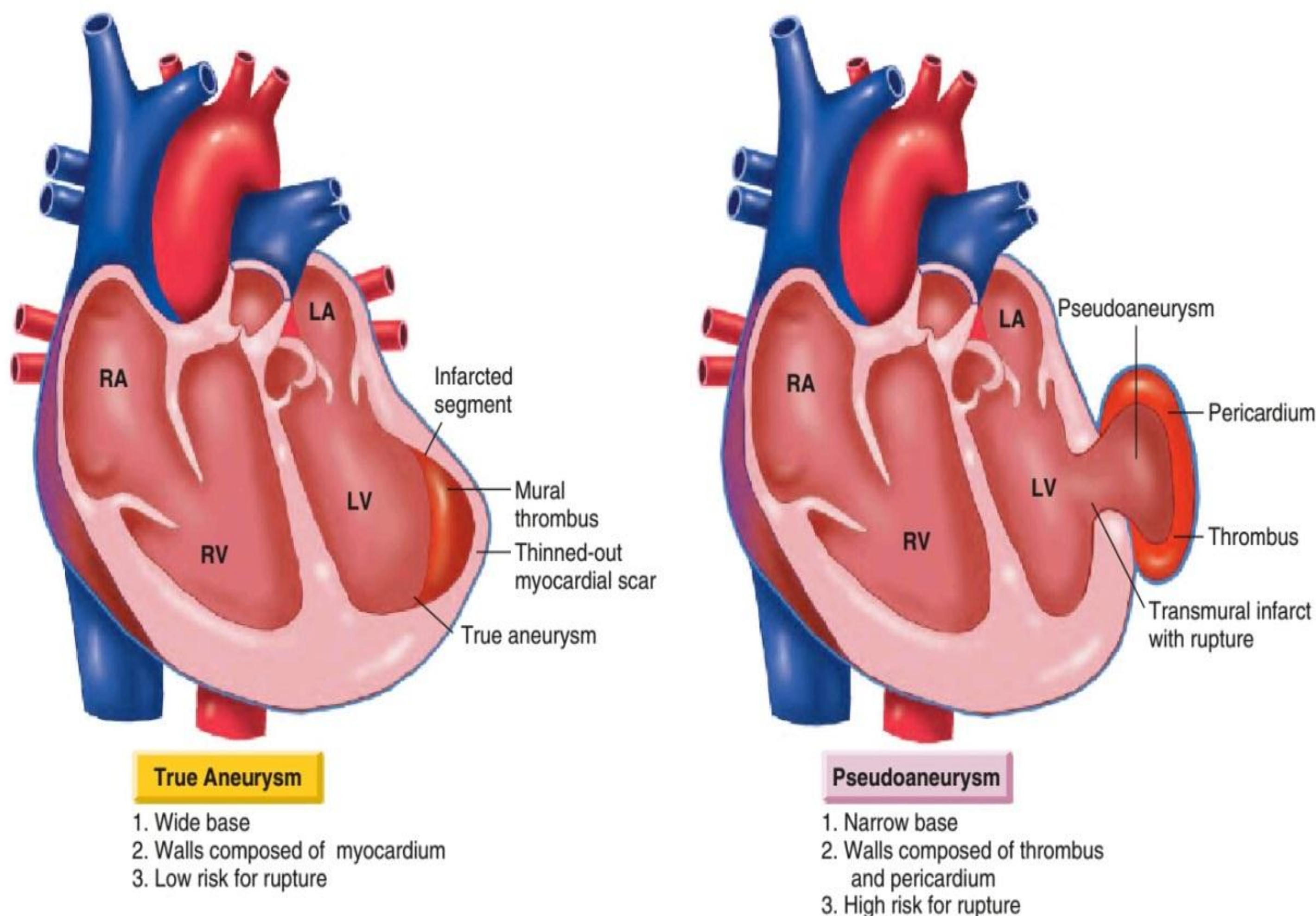
ABG and Lactic acid should be trended every 1-6 h initially to assess response to initial resuscitation.

Patient with acute RVF or LVF suspected ischemic etiology should undergo immediate cardiac catheterization.

Diagnosis

Spurious estimation of LV end diastolic pressure based on measurement of PACWP can occurring patient with marked MR tall V wave in left atrial pressure
MR, VSD ,ventricular aneurysm and pseudoaneurysm must be excluded before making the diagnosis of C S
Mechanical complication should be suspected in any patient with STEMI and if it is present with cardiogenic shock need immediately evaluation :

1. **Hemodynamic**
2. **Angiographic**
3. **echocardfiographic**



EFIGURE 59.8 Differences between a pseudoaneurysm and a true aneurysm. *LA,* Left atrium; *LV,* left ventricle; *RA,* right atrium; *RV,* right ventricle. (From Shah PK: Complications of acute myocardial infarction. In Parmley W, Chatterjee K, editors. Cardiology. Philadelphia: Lippincott; 1987.)

Medical management

Caused by impaired LV function , inotrop can provide pharmacological support to maintain MAP and augment cardiac output

Inotropes should be administrated at the lowest dose Inotropes :

Improving hemodynamics

Do not appear improve survival

Vasodilators

Increase cardiac output

Reduce LV filling pressure

Reduce coronary Perfusion pressure and decrease cardiac function

Stabilization and resuscitation strategy

Intravenous fluid is clinical challenge in early management of CS as difficult to be assess

Echocardiography right sided heart volume and R/O pericardial collection

The definite method of volume status assessment is right heart catheterization

Oxygenation and ventilation

Pulse Oximetry should be used

In acute care setting blood O2 saturation more than 90% are accepted

Vasopressor support

Receptor activity:

***Alpha 1 receptor**

increase smooth muscle contraction

Vasoconstriction

Increase SVR

***Beta 1 receptor**

Increase heart rate

Increase myocardial contractility

***B2 receptor**

Smooth muscles relaxation

Decreased SVR

Table 2. Summary of Systemic Vasopressors

Agents	Mechanism	Effect	Indications	Considerations
Phenylephrine	A1 agonist	Vasoconstriction	Various forms of shock	Caution in cardiac dysfunction as it increases afterload
Norepinephrine	A<B agonist	Inotropy, chronotropy, dromotropy, and vasoconstriction	Most common first line agent in shock	Most benefits demonstrated in septic shock
Epinephrine	A≪B agonist	Inotropy, chronotropy, dromotropy, and vasoconstriction	Commonly used as second line agent or first line in anaphylactic shock	Surviving Sepsis Guidelines has most data for epinephrine as second line agent
Dopamine	Dose dependent A, B, and D agonism	Inotropy, dromotropy, chronotropy, and vasoconstriction (at highest doses)	Second line agent in most forms of shock	SOAP II trial demonstrated more incidence of tachy-arrythmias and increased mortality in CS patients when dopamine was used as first line
Vasopressin	V1 agonist	Vasoconstriction	Second line agent in most forms of shock	On or Off dosing, can cause hyponatremia
Dobutamine	B agonist	Inotropy and mild vasodilation	Commonly used in cardiogenic shock	May contribute to hypotension
Levosimendan	Myofilament Ca^{2+} sensitizer and K^{+} channel modifier	Ionotropy and inodilator	Used in acutely decompensated chronic heart failure	Minimal effect on myocardial oxygen consumption

CS indicates cardiogenic shock; SOAP, Sepsis Occurrence in Acutely Ill Patients.

Continuous renal replacement therapy should be in :

Stage 2 K I defined by :
Increase serum cr>2× baseline
U O <0,5 ml /kg/h for 12h
And life change in fluid, electrolytes ,
acid _base balance need for dialysis

Hemodynamic monitoring

Goals:

- **Produce stable vital singe**
- **Adequate tissue perfusion**

Continuous BP monitoring

Telemetry

Pulse oximetry

Temperature

Respiratory rate

Input and output

Pulmonary artery catheter for identification of patient requiring mechanical support

Continuous hemodynamic monitoring and measurement of

1. Fluid states
2. Central venous oxygen saturation
3. Response to therapy
4. Indicate the effectiveness of ventricular support

PAC use does not confer a mortality benefit or reduce the length of intensive care unit or hospital states

In critical ill patient increase mortality

Complication of PAC

1. **Arrhythmias.**
2. **Heart Block.**
3. **Infection.**

LBBB contraindicated

MCS Device:

1.IABP in patient with STEMI with hemodynamic instability who require support for bridging for PCI or surgical correction
2.Those with cardiogenic shock and dose not responded to medical management.
3. Those with refractory ischemia not alleviated by anther treatment
IABP - shock II trail 600patient were randomized to either IABP or medical l treatment ,no difference was seen in the primary end point of 30 day and follow up in 12 month all cause of mortality.
4. axial flow pumps(impella LP 2.5 , impella CP).
5. Left atrial - to femoral arterial ventricular assist devices (tandam heart)ز
6. ECMO .

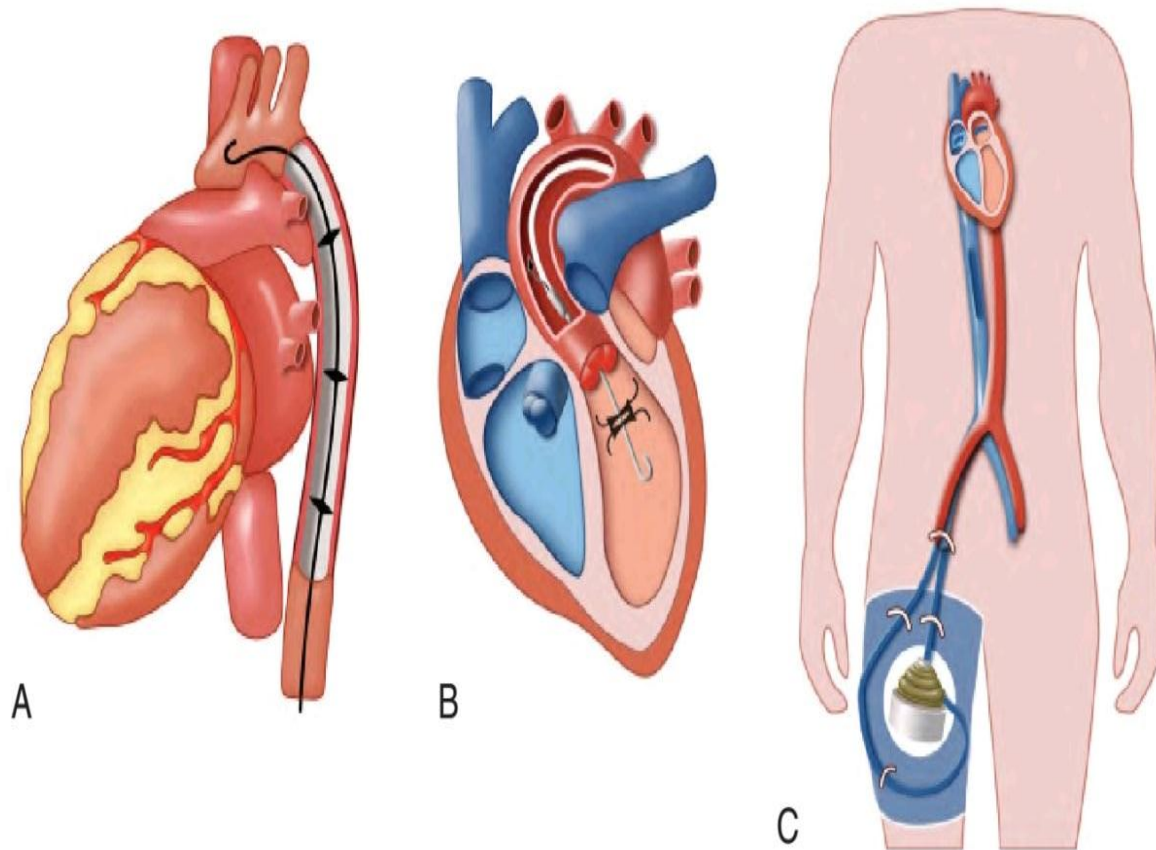


FIGURE 59.22 Schematic representation of examples of major categories of nonsurgical mechanical circulatory support. **A,** Intra-aortic balloon pump inserted into the descending aorta between the arch vessels and renal arteries. **B,** Impella Recover (Abiomed, Aachen, Germany). This rotational flow device is percutaneously inserted through the femoral artery and positioned across the aortic valve, with flow intake in the left ventricle and outflow in the aorta. **C,** TandemHeart (CardiacAssist, Pittsburgh). A cannula is inserted percutaneously through the right femoral vein and advanced toward the right atrium, where it is introduced by transatrial septal perforation, to establish inflow into an external rotational motor. A cannula in either femoral artery then provides the outflow. (Modified from Desai NR, Bhatt DL. Evaluating percutaneous support for cardiogenic shock: data shock and sticker shock. Eur Heart J 2009;30:2073.)

Revascularization

Five therapies frequently used in cardiogenic shock

1. **Inotropes.**
2. **MCS.**
3. **Fibrinolysis.**
4. **PCI.**
5. **CABG.**

Revascularization improve survival

SHOCK trail show at 30 days mortality rate was 46.7% in revascularization group

In medical treatment group 56%

THANK YOU